

## Original Article

# Electrophysiological characteristics of paroxysmal atrial fibrillation originating from superior vena cava: a clinical analysis of 30 cases

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**Abstract:** To analyze characteristics of electrocardiogram (ECG), electrophysiological intracardiac mapping and radiofrequency ablation (RF) of paroxysmal atrial fibrillation (PAF) originating from superior vena cava (SVC), aiming to investigate electrophysiological characteristics of PAF with SVC origin. Clinical data of 30 subjects (18 men and 12 women, aged,  $58.6 \pm 15.5$  years) with PAF of SVC origin were retrospectively analyzed; All patients underwent RF during 2006.9-2012.7. ECG of AF and atrial premature contractions (APCs), procedure and fluoroscopic time, numbers of ablation sites within SVC, complications and success rate were studied. Compared with P wave of sinus rhythm (SR), APCs of SVC origin exhibited higher amplitude in lead II ( $0.23 \pm 0.11$  vs.  $0.15 \pm 0.06$  mv), III ( $0.19 \pm 0.09$  vs.  $0.13 \pm 0.08$  mv), AVF ( $0.21 \pm 0.13$  vs.  $0.14 \pm 0.10$  mv), V<sub>2</sub> ( $0.24 \pm 0.07$  vs.  $0.15 \pm 0.09$  mv) and V<sub>3</sub> ( $0.21 \pm 0.09$  vs.  $0.12 \pm 0.05$  mv) ( $P < 0.05$ ), as well as more biphasic polarity in lead V<sub>1</sub> (80.0% vs. 26.6%,  $P < 0.05$ ) and isoelectric in AVL (60.0% vs. 6.7%,  $P < 0.05$ ). In terms of left pulmonary vein (LPV) and right pulmonary vein (RPV) electrical isolation, procedure time ( $14.3 \pm 11.5$  vs.  $33.7 \pm 14.2$ ,  $28.1 \pm 6.8$  min,  $P < 0.05$ ), fluoroscopic time ( $9.6 \pm 3.8$  vs.  $21.1 \pm 9.3$ ,  $19.4 \pm 9.7$  min,  $P < 0.05$ ), ablation sites ( $11.2 \pm 3.1$  vs.  $37.1 \pm 13.7$ ,  $31.4 \pm 10.4$  points,  $P < 0.05$ ) of SVC isolation (SVCI) remarkably decreased compared with that of mean LPV and RPV. After the procedure, 9 patients still presented paroxymal rapid firing within the SVC in the setting of SR restoration, 2 patients developed paroxysmal atrial flutter within 1 month after completion of ablation and were controlled by antiarrhythmic drugs. The APCs and AF of SVC origin manifested distinctive ECG features, which could be helpful to distinguish SVC from other foci before ablation, the completion of SVCI required shorter procedure and fluoroscopic time, as well as less ablation points, and meanwhile, the success rate was high with less complication.

**Keywords:** Atrial fibrillation, superior vena cava, radiofrequency ablation, electrophysiological characteristics

## Introduction

Atrial fibrillation (AF) is one of the most common arrhythmia in clinical practice, most of which triggered by ectopic foci originating from pulmonary veins (PVs) [1]. Based on this finding, ablation procedures for AF have become an established and increasingly used option for managing patients with symptomatic arrhythmia, circumferential pulmonary veins isolation (CPVI) is considered to be the most effective treatment of AF [2]. However, the long-term results of this procedure remains unsatisfactory [3] partly due to some ectopic foci in the non-PVs areas. It was reported that 6-12% of paroxysmal AF (PAF) originating from superior vena cava (SVC) [4], 20% recurrent AF after CPVI arising from SVC [5], meanwhile, SVC accounted for

37% of non-PVs originating AF, which supported that SVC played a very important role in AF initiation. The aim of this study was to investigate ECG features of PAF and atrial premature contractions (APCs) of SVC origin (SVC-APCs), character of intracardiac mapping and SVC isolation (SVCI), and to evaluate the efficacy of SVCI, which could be helpful to identify SVC as the target vein before procedure and improve success rate of AF ablation.

## Material and methods

### Subjects

We retrospectively analyzed 136 PAF cases receiving radiofrequency ablation (RF) during 2006.9-2012.7, of whom 30 patients (18 men

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**Table 1.** Baseline characteristics of patients undergoing radiofrequency ablation

Characteristics	Values
Age (years)	58.6 ± 15.5
Men (%)	18 (60.0%)
LA diameter (mm)	37.5 ± 2.8
LVEF (%)	55.6 ± 6.4%
AF duration (months)	30 ± 16
CHA2DS2-VAS <sub>c</sub> score	2.5 ± 1.5
Comorbidities	
Hypertension (n)	12 (40.0%)
Coronary artery disease (n)	6 (20.0%)
OSAHS (n)	6 (20.0%)
Mellitus (n)	10 (33.3%)
Stroke (n)	2 (6.7%)
AAD Treatment	
Amiodarone (n)	20 (66.6%)
Propafenone (n)	10 (33.3%)
Beta blockers (n)	18 (60.0%)
Ibutilide (n)	2 (6.7%)
Sotalolol (n)	4 (13.3%)

and 12 women, aged, 58.6 ± 15.5 years) were confirmed with PAF of SVC origin. Patients with hyperthyroidism, old myocardial infarction, rheumatic valve disease, chronic obstructive pulmonary disease (COPD) were not recruited in this study. All patients had clinically documented APCs initiating symptomatic AF refractory to at least 2 kinds of anti-arrhythmic drugs. Some subjects suffered hypertension, coronary artery disease, diabetes mellitus and obstructive sleep apnea hypopnea syndrome (OSAHS). 2 cases had history of stroke. Underlying structural heart diseases were ruled out in all patients by screening of chest X-ray and echocardiography. Baseline characteristics of patients were shown in **Table 1**. None had undergone any prior AF ablation. Transesophageal echocardiography was performed to exclude thrombi in the left atrium (LA).

### *Electrocardiogram analysis*

P waves were analyzed by two blinded physicians on 12-lead ECG recorded at a paper speed of 50 mm/s and amplitude of 0.5 mv/cm, clearly visible P waves or distinctive P waves superimposed on the preceding T waves were analyzed and classified as positive, negative, isoelectric or bi-phasic. Ectopic P wave morphology of APCs was compared with that of

sinus rhythm in 12 leads. Subtraction was used to identify the polarity and amplitude of P wave buried in prior T wave.

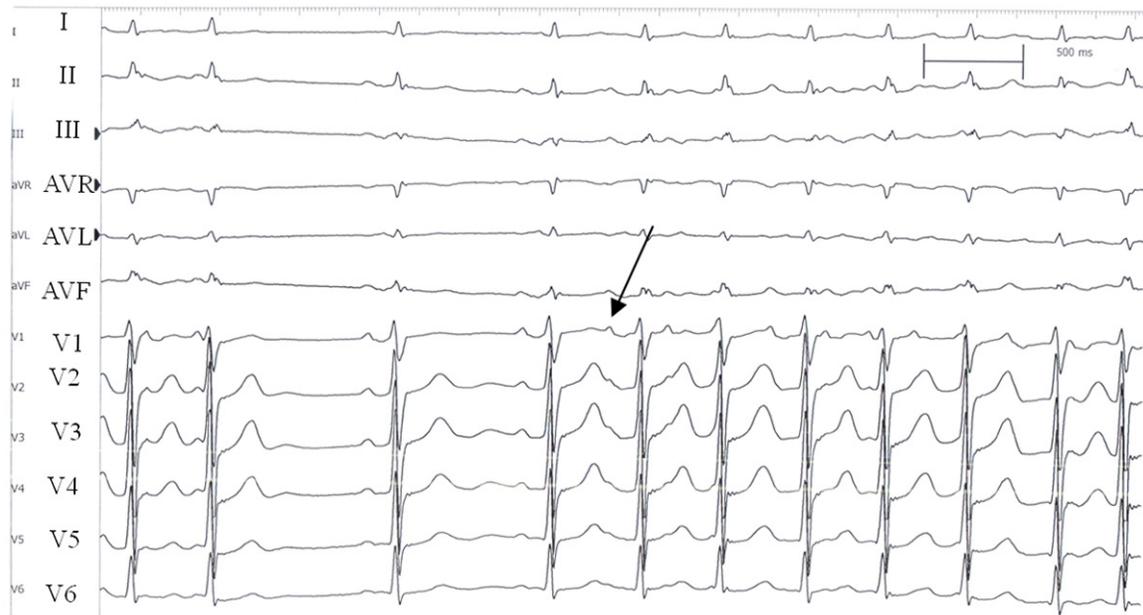
### *Electrophysiological study*

All patients were kept on oral anticoagulation with warfarin 1 month prior to procedure, warfarin was withdrawn 3 days before ablation, all anti-arrhythmic drugs were discontinued at least five half-lives, amiodarone was discontinued at least 6 weeks before procedure. Every patient after giving informed consent underwent an electrophysiological study in a fasting and conscious-sedated state. Intracardiac electrograms were recorded using an electrophysiology system (Prucka CardioLab™ General Electric Health Care System Inc, USA). One decapolar mapping catheter (Biosense Webster, Diamond Bar, CA) was positioned in the coronary sinus (CS) through the right jugular vein access, using the standard Brockenbrough technique [6], atrial transeptal puncture was performed under fluoroscopic guidance and two L<sub>1</sub>-type Swartz sheathes (St Jude Medical, Minneapolis, MN) were transeptally introduced into LA via right femoral vein. Intravenous unfractionated heparin 5000 U was administered immediately after atrial transeptal puncture and followed 1000 U/h to maintain an activated clotting time (ACT) of 300-350 S, the ACT level was monitored every 30 min. In addition, heparinized saline solution was continuously infused through the transeptal sheath (3 mL/min) to avoid formation of thrombi or air emboli. Selective PV venography was performed to identify all PV ostia. One decapolar circular mapping catheter (Lasso, Biosense Webster) was placed at the ostium of each PV to record PV potentials.

### *Circumferential pulmonary vein isolation (CPVI)*

The procedure of CPVI was performed under the guidance of CARTO system (Biosense Webster), a 3.5 mm saline-irrigated catheter (Navi-star, Thermocool, Biosense Webster) was transeptally advanced into LA via Swartz sheath, LA geometry was reconstructed and each PV ostium was tagged on it. Pulmonary vein antral isolation was performed 5 to 10 mm outside of the PV ostia. Ipsilateral pair of left and right PVs was isolated in one circumferential lesion. RF current was delivered point by point at a target temperature 43°C, maximum

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**Figure 1.** ECG characteristics of AF with SVC origin (arrow indicated), the morphology of SVC-APC was identical to P wave of SR, positive polarity with higher amplitude in inferior leads and biphasic pattern in lead V1 (patient 1).

**Table 2.** Comparison of ECG characteristics between SVC-APCs and sinus rhythm

P Wave (lead)	SVC-APCs	SR
Amplitude of II (mv)	0.23 ± 0.11*	0.15 ± 0.06
Amplitude of III (mv)	0.19 ± 0.09*	0.13 ± 0.08
Amplitude of AVF (mv)	0.21 ± 0.13*	0.14 ± 0.10
Amplitude of V <sub>2</sub> (mv)	0.24 ± 0.07*	0.15 ± 0.09
Amplitude of V <sub>3</sub> (mv)	0.21 ± 0.09*	0.12 ± 0.05
Biphasic pattern of V <sub>1</sub> (n)	24 (80.0%)*	8 (26.6%)
Isoelectric of AVL (n)	18 (60.0%)*	2 (6.7%)

SVC-APCs: SVC originated APCs, SR: sinus rhythm, \* $P < 0.05$  SVC-APCs vs. SR.

power of 35 W (Stocker generator, Biosense Webster Inc, Diamond Bar, CA, USA), and an infusion rate of 17 mL/min. The end point of CPVI was electrical isolation of PV potentials, which was confirmed by Lasso catheter mapping during sinus rhythm or isoproterenol infusion after 30 min. The end point was bidirectional conduction block between LA and PVs. If AF was not terminated by above-mentioned ablation, additional ablation lines were created, including a bottom line connecting both inferior PVs and/or a mitral isthmus line. Linear ablation at cavotricuspid isthmus was created in patients with documented or inducible cavotricuspid isthmus dependent atrial flutter, until bidirectional isthmus block was achieved.

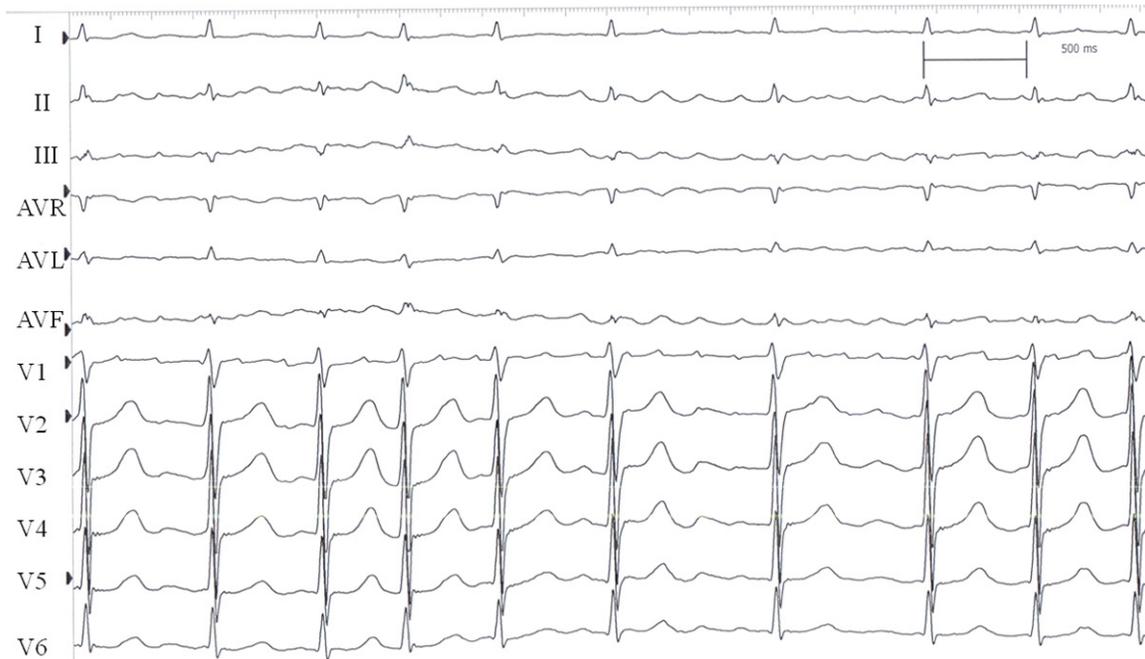
### Superior vena cava mapping and isolation

After the successful completion of CPVI, if APCs still existed and PAF was initiated by isoproterenol infusion, SVC mapping was subsequently performed. Circular mapping catheter was positioned at the level of the lower border of the pulmonary artery above the SVC- right atrium (RA) junction guided by SVC venography. RF current was applied at the point with SVC potentials for 30 s using 3.5 mm saline-irrigated catheter with maximum temperature set at 50°C and maximum power at 35 W. At the posterior-lateral wall of SVC, high output pacing (10 mA) was performed at each point before RF delivery. If diaphragmatic stimulation was positive at this site, ablation was avoided to prevent phrenic nerve injury [7]. As RF delivery at the anterolateral free wall of SVC, low energy with maximum temperature 40°C and maximum power 30 W was applied to sites adjacent to sinus node (SN) with the purpose of preventing SN injury. The end point was disappearance of SVC potentials or dissociation of SVC potentials with RA electrical activities.

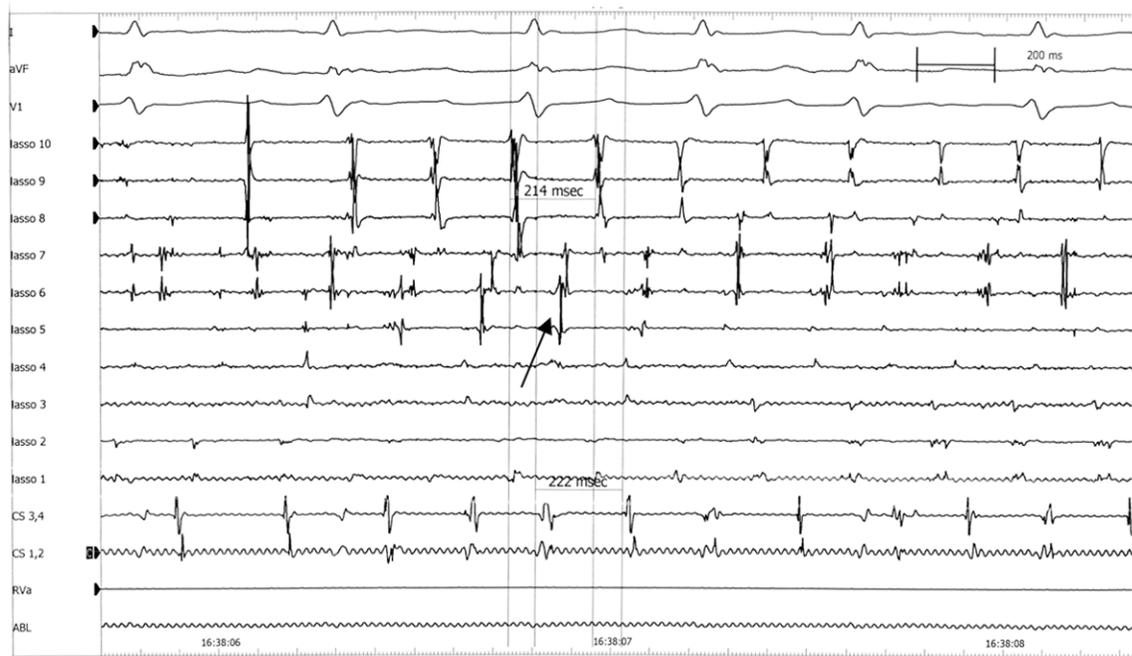
### Postoperative care and follow up

After the procedure, oral anticoagulation with warfarin was resumed and the international

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**Figure 2.** The morphology of AF wave was similar to P wave of SR and SVC-APC (patient 1).

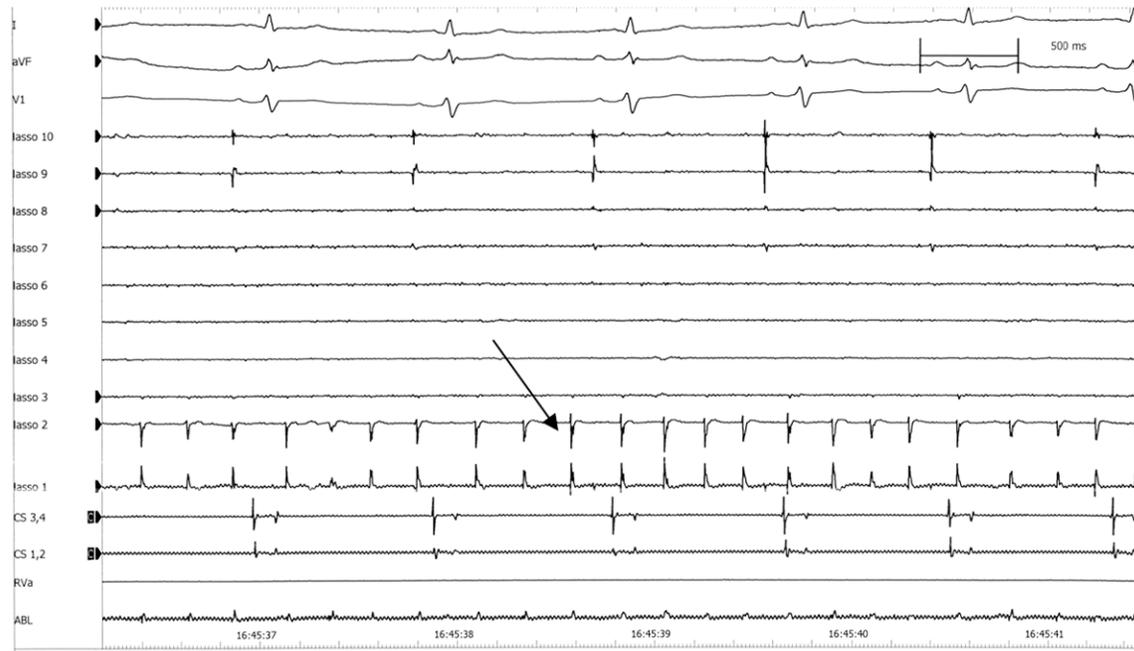


**Figure 3.** SVC potentials (arrow indicated) could be recorded by circular catheter (Lasso 5-7) within SVC in intracardiac mapping, the earliest activation obtained at Lasso 5, no SVC potentials appeared at other sites of circular catheter, which suggested local electrical connection confined to the sites of Lasso5-7 (patient 3).

normalized ratio (INR) was controlled in the therapeutic range (2.0-3.0), warfarin was continued at least 3 months, meanwhile, anti-

arrhythmic drugs were not recommended for patients. Follow up was at two weeks, 1, 3, 6, 9, 12, 18, 24 month after procedure at outpatient

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**Figure 4.** ECG and intracardiac mapping after successful ablation: SR was restored in surface ECG after ablation, irregular rapid firing (arrow indicated) was recorded by the distal circular catheter and ablation catheter in SVC, meanwhile, SR recorded by the proximal circular catheter in HRA, indicating SVC-HRA exit block. HRA: high right atrium (Lasso: circular catheter; CS: coronary sinus; RVa: right ventricular apex; ABL: ablation catheter) (patient 3).

**Table 3.** Comparison of PV and SVC isolation

	SVC	Mean LPV	Mean RPV
Procedure time (min)	14.3 ± 11.5*	33.7 ± 14.2	28.1 ± 6.8
Fluoroscopic time (min)	9.6 ± 3.8*	21.1 ± 9.3	19.4 ± 9.7
Ablation sites (n)	11.2 ± 3.1*	37.1 ± 13.7	31.4 ± 10.4
Number of potential (n)	3.3 ± 1.2*	7.8 ± 1.5	7.3 ± 1.4

LPV: left pulmonary vein; RPV: right pulmonary vein, VC: superior vena cava, \* $P < 0.05$  SVC vs. Mean LPV, Mean RPV.

clinic using 24-h Holter monitoring for all patients. Recurrence was defined according to patient's symptom, and/or if arrhythmia lasting more than 30 s was documented. 3-month after the ablation was considered a blanking period, any AF recurrence within this period was not diagnosed as ablation failure.

### Statistical analysis

Continuous variables were expressed as mean ± SD and categorical variables as proportions (%). Data were analyzed with unpaired  $t$ -test; categorical variables were compared with  $\chi^2$  tests. A value of  $P < 0.05$  was considered statistically significant. All analyses were performed using SPSS 13.0.

## Results

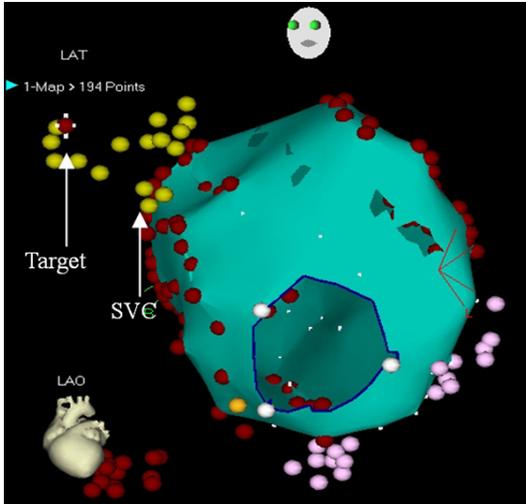
### Electrocardiographic features of PVCs and AF originating from SVC

The morphology of SVC-APCs was similar to those of sinus P waves (**Figure 1**), which demonstrated positive polarity in lead I,

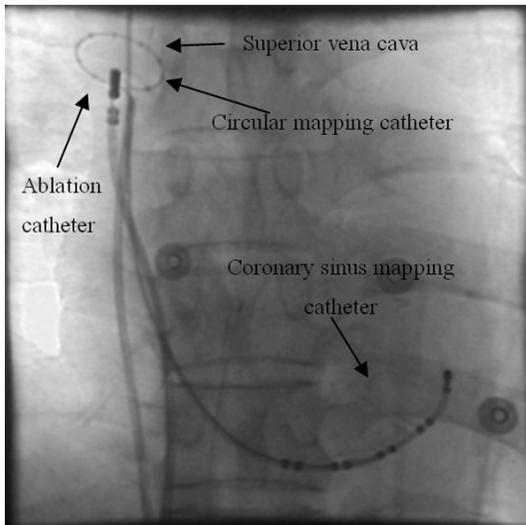
II, III and AVF, negative polarity in AVR, more frequency of biphasic pattern in lead  $V_1$  and iso-electric in AVL, the first component was positive and the second was negative (+/-) in  $V_1$ . Compared with P waves of sinus rhythm (SR), SVC-APCs exhibited higher amplitude in lead II, III, AVF,  $V_2$  and  $V_3$  (**Table 2**). Based on polarity and morphology in 12 leads, fibrillation wave of AF with SVC origin was identical to P wave of SR and SVC-APCs in the presence of AFCL prolongation in 9 cases (**Figure 2**).

### Acute procedure result

120 PVs and 30 SVCs were targeted in 30 patients; bottom line and cavotricuspid line were created in 14 and 11 cases respectively.



**Figure 5.** Successful ablation site in CARTO mapping system (LAO projection), AF was converted into SR just after the ablation of target site located in the anterior-lateral aspect of SVC. LAO: left anterior oblique (patient 1).



**Figure 6.** The position of circular mapping catheter and ablation catheter within SVC in the X-ray (PA projection). PA: posterior anterior (patient 1).

All patients had frequent PACs and inducible AF after CPVI alone, and SVC potentials could be recorded by circular catheter within SVC (**Figure 3**). Compared with extensive pulmonary potentials recorded by circular catheter, SVC potentials were less and detected on confined area (**Figure 4, Table 3**). In 5 subjects, AF converted into organized atrial tachycardia after the completion of CPVI. SVCs isolation was achieved under the guidance of CARTO system (**Figure 5**)

and X ray (**Figure 6**) in all subjects. Burst rapid foci activity within SVC still remained in 9 subjects in the presence of sinus rhythm restoration after SVCI (**Figure 4**). In order to compare SVC with single pulmonary vein ablation, mean LPV and RPV procedure and fluoroscopic time as well as ablation sites (half of ipsilateral LPV and RPV ablation respectively) were calculated, the above-mentioned index of SVCI remarkably decreased when compared with that of mean LPV and RPV (**Table 3**). No complications, including pericardial effusion, phrenic nerve and sinus node injury were observed.

#### Follow-up

All subjects underwent at least 12 months follow-up (mean  $18.3 \pm 6.3$  months), 2 cases presented paroxysmal atrial flutter at 14 and 23 days after completion of RF ablation respectively, which were well controlled by orally administration of propafenone (150 mg, 1/8 h), the therapeutic duration was 1 month. After 3 months of blanking period, no patients took any anti arrhythmic drugs. The rest of patients were free of atrial arrhythmia during the follow-up. All subjects were continuously monitored after completion of this study, 1 patient was confirmed recurrence 10 months after ablation and underwent another procedure.

#### Discussion

The present study demonstrated some electrophysiological characteristics of AF originating from superior vena cava (SVC-AF), which revealed clinical features different from other sites originated AF, at the same time, this study indicated SVC played an very important role in AF initiation, and relative high success rate of electrical SVC isolation (SVCI) under the guidance of CARTO system without major complications. In our study SVC-AF accounted for 22% of AF cases undergoing conventional mapping and ablation. These findings suggested routine SVC mapping should be applied for those with recurrent AF after procedure of CPVI or suspected SVC-AF based on ECG features.

We carefully analyzed documented ECG of atrial premature contractions (APCs) arising from SVC (SVC-APCs), in general, which was similar to the morphology of sinus P wave because their foci were in close proximity to each other, such as positive polarity in inferior leads and negative in AVR. However, due to its relatively

anatomical superior position, SVC-APCs manifested some distinctive characteristics. This study showed P wave amplitude was greater than that of SR in inferior and  $V_2$ ,  $V_3$  leads, more isoelectric pattern in AVL, in our study, 16 foci of SVC presented this morphology, however, only 2 SR P waves had this character. Meanwhile, high frequency of biphasic pattern in lead  $V_1$  was detected in this study, which was consistent with previous study [8-10], however, the underlying mechanism of this phenomenon remain unclear, the relative position of ectopic beats within SVC to lead  $V_1$  were different, most of SVC-APCs were relatively leftward to the sinus node, the initial and final vector reflection on lead  $V_1$  may exhibit positive and negative respectively, which could explain the biphasic pattern. SVC is anatomical close to the right superior pulmonary vein (RSPV), therefore, P wave polarity of SVC tachycardia is identical to that of RSPV tachycardia [11], sometimes RSPV originating tachycardia was misdiagnosed as sinus node or high right atrium and SVC origin [12, 13]. It was [11, 14] reported that a notched P wave in lead II with positive polarity in lead  $V_1$  was helpful in predicting RSPV origin. Our study also demonstrated that all SVC-APCs did not present notched figure of P wave in lead II, which could distinguish an arrhythmogenic focus of AF from SVC or RSPV. In addition to ECG pattern, most of LSPV originating tachycardia manifested on and off character (initiated and terminated more than 10 times per day), this phenomenon was not common in AT of SVC origin [14]. During the episode of AF, morphology of AF wave was similar to that of SVC-APCs and sinus P wave in 9 subjects, especially in the period of AFCL prolongation, which suggested AF sometimes was directly driven by SVC foci activation and intermittently manifested as one form of arrhythmia identical to atrial tachycardia. Kuo also reported this phenomenon in his study [15].

If morphology of APCs resembled of those arising from SVC, SVC was therefore preferentially mapped and isolated after CPVI, patients were monitored 30 minutes, if APCs disappeared and AF could not be reinduced by isoproterenol infusion and rapid stimulation from coronary sinus (CS), then we determined it as SVC-driven AF, simultaneously within the period of 30 minutes observation, we mapped inferior vena cava and CS by lasso catheter, which did not detect any ectopic activity in these areas, how-

ever marshall ligament and crista terminalis (CT) were not routinely checked, but disappearance of APCs after SVCI and non-inducibility of AF did not support origin of these areas.

Compared with each pulmonary vein isolation, complete SVCI required less ablation sites, total procedure and fluoroscopic time, which suggested myocardial connections between SVC and right atrium were less than that of pulmonary vein-left atrium, our study indicated SVC potentials merely could be mapped in local area of SVC, however, potentials extensively existed around pulmonary vein. Many studies [16] have confirmed this finding. In present study, rapid firing still could be recorded within SVC in the setting of sinus rhythm restored after SVC isolation in 9 cases, most of which exhibited as irregular cycle length and favored focal substrate, the underlying electrophysiological mechanism could be increased automaticity or triggered activity [17, 18]. During the process of CPVI, we observed that AF transformed into organized rapid atrial arrhythmia in 5 subjects, which indicated SVC arising AF could conduct into left atrium by a fibrillated manner, and left atrium was not an indispensable component for maintenance of AF arising from SVC.

In our study, 2 patients suffered from paroxysmal atrial flutter within the first month After the procedure, ECG revealed low amplitude in inferior leads and positive polarity of flutter wave in lead  $V_1$  and  $V_2$ , the etiology could be attributable to scar-dependent reentry within left atrium based on ECG findings. After orally administration of propafenone, the ablation related atrial flutter was significantly inhibited. No major complication was found in total 30 subjects, it was reported that right phrenic nerve and sinus node injury were major complications of SVCI, especially at the time of delivering RF energy at the free wall of SVC, in this study, we prophylactically performed high output pacing at each site before ablation at the posterolateral free wall and low energy delivery to sites at the anterolateral free wall, Chen et al. [19] reported that sinus node damage mainly occurred during ablation within the anterolateral free wall of SVC, the definition of the junction of right atrium and SVC is very important, ablation close to SVC orifice should be avoided due to anatomical adjacent position of sinus node. When we did SVC mapping, the geometry of right atrium

was not created, venography of SVC in combination with circular catheter mapping at the junction of SVC- right atrium determined the precise border of SVC- right atrium, and then we can locate the site of ablation within SVC. It was confirmed that these measures could effectively prevent the occurrence of above-mentioned complications. During 24 months follow-up, only one patient suffered from AF recurrence and underwent another procedure of SVCI, which demonstrated two reconnections at the anterolateral wall of SVC. Some studies [20] revealed that 5 years success rate of SVCI after a single ablation procedure could reach 73%, and SVCI without CPVI is an acceptable therapeutic strategy in patients with AF originating from the SVC only, however, in this study, we could not completely rule out the role of PVs in AF initiation, therefore, CPVI plus SVCI was performed in all subjects.

After SVC isolation, burst rapid firing activity still could be detected within SVC in 9 patients; we carefully measured cycle length of these rapid firing which varied beat by beat, this finding suggested the mechanism could be increased automaticity rather than reentry, however, other study demonstrated focal reentry could be underlying mechanism of atrial tachycardia with SVC origin [21, 22]. In our study, male patients accounted for 60% of total cases, because this was a small scale clinical research, only 30 cases were identified as SVC origin from 136 paroxysmal AF patients, the small sample could partially explain the gender difference.

Our research confirmed that SVC was one of the most important non PVs origins of AF, and SVC should be targeted in order to decrease the recurrence of AF after ablation. Although ectopic foci arising from PVs are the predominant sources for the initiation and maintenance of AF in a majority of cases, and CPVI remains corner stone of AF ablation, additional SVC isolation should be considered if AF recurred after simple CPVI or suspected SVC origin. However, routine prophylactic SVCI for all AF patients still remain controversial because the procedure could result in severe complication, and it is recommended that SVCI should be performed only if SVC focus is recognized [23]. For patients with SVC related AF, SVCI alone may successfully eliminate AF with relative simple procedure and less X-ray exposure time without the need

of CPVI. The main limitations of this study were small number of subjects and relative short follow-up period.

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### Disclosure of conflict of interest

None.

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